ON THE

MINUTE ANATOMY AND PATHOLOGY

OF

BRIGHT'S DISEASE OF THE KIDNEY,

AND ON THE

RELATION OF THE RENAL DISEASE TO THOSE DISEASES OF THE LIVER, HEART, AND ARTERIES, WITH WHICH IT IS COMMONLY ASSOCIATED.

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There is, perhaps, no single disease which has excited more interest among all classes of the profession, than that form of renal degeneration, the existence of which was first made known by Dr. Bright, and which has been named after that distinguished physician.

In all that relates to the clinical study of Bright's disease, the labours of Dr. Bright, and of his followers, have been attended by an amount of success almost unprecedented in the history of any other disease. Pathologists, however, have not as yet succeeded in obtaining a knowledge of the precise nature of those changes which the kidney undergoes in the course of this disease. This want of success may, perhaps, be attributed to the fact, that those who have entered upon the investigation, have done so with a certain preconceived notion on the subject, which has induced them

to direct their attention too exclusively to the vascular system of the gland, while they have neglected the study of other portions of the organ, which, there seems reason to believe, are primarily and essentially concerned in the disease; the parts to which I refer are the urinary tubules, with their lining of secreting, or epithelial, cells.

I have ascertained that the epithelial cells of the healthy kidney contain a minute quantity of oil, in the form of yellowish, highly refracting globules, the appearance of which in the cells of the liver is well known to microscopical observers. The quantity of fat in the cells of the healthy kidney is much less than in those of the healthy liver, but I have found it present, in greater or less quantities, in the healthy kidneys of more than twenty subjects which I have examined for this point, since my attention was first directed to it.

In every healthy kidney there are many cells entirely free from oil (see Plate I. fig. 1), while others contain only one or two minute particles (figs. 2 and 3); others, again, contain several scattered over the interior of the cell (fig. 4). The quantity in different kidneys varies greatly within the limits of health, and I have generally observed that when the liver cells contain a more than ordinary amount of fat, the kidney cells in the same subject present an unusual quantity of the same material. In two or three cases, the quantity of fat in the kidneys has been extremely small, but never entirely absent.

The secreting cells of the kidney, then, resemble those of the liver, in containing a certain proportion of oil, and the presence of this material in such a situation appears to indicate that a certain quantity of fat is excreted by the kidneys, as well as by the liver.

Having premised thus much of the healthy epithelium, with its contents, Bright's disease may be described as *primarily and essentially an exaggeration of the fatty matter which exists naturally in small quantities in the epithelial cells of the healthy organ.* A specimen of the disease in an

advanced stage, examined with the microscope, presents epithelial cells in every degree of engorgement, from the incipient enlargement of the particles in fig. 5, in which the cell nucleus is still visible, to the complete engorgement of the cell in figs. 6, 7, and 8, in which the nucleus is concealed by the fatty globules.

The disease, then, appears to be a fatty degeneration of the kidney, precisely analogous to the fatty degeneration of the liver.

It will scarcely be necessary to remind the Society, that about four years since, Mr. Bowman* discovered that in fatty degeneration of the liver, the fat is contained in the secreting cells of the organ; that it is, in fact, an increase of those fatty globules, the existence of which, in small quantities, in the cells of the healthy gland, had been previously discovered by Henle and Mr. Erasmus Wilson.

Returning to the kidney. If a portion of the diseased gland be torn up with needles, and examined with a magnifying power of about 400 diameters, the gorged tubes are seen presenting the appearance represented in fig. 9. In examining a section of the cortical portion of the kidney with a power of 100 diameters, sets of convoluted tubes are seen crowded with their fatty contents. A set of gorged tubes, presenting itself either on the surface of the gland, or on the surface of a section, would constitute one of the so-called "granulations of Bright." So that, by examining a section by a low power, it is ascertained that the deposit is contained within the tubes; and by breaking up the tubes and examining them with a higher power, we arrive at a knowledge of the fact, that the fatty material is contained within the epithelial cells which line the tubes.

The presence of oil globules in a free state, or in any other situation than in the tubes, is accounted for by the rupture of the cells and tubes, which probably is a frequent occurrence, in consequence of their over-distension by the accumulated fat.

* Lancet, January 1842.

The accumulation does not take place simultaneously and equally in every part of the tubes. Those portions of the tubes which form the pyramids, and which are lined by epithelium, having more the character of the epithelium lining excretory ducts, than that of the true glandular epithelium, these portions of the tubes, with their lining epithelium, do not become gorged in any great degree, except in cases where the disease has been of long duration, and in which the cortical portion of the kidney has become wasted. In these cases, the epithelium of the cones becomes gorged with fat; perhaps, in consequence of its assuming, in some degree, the function of glandular epithelium, to compensate for the waste of the proper secreting portion of the gland.

Another portion of the tubes, which, according to my observations, seldom contains much fat, is the expanded portion, which, as Mr. Bowman has shown, forms the investment of the Malpighian plexus. Mr. Bowman describes the epithelium of this expanded portion of the tube as extremely delicate, and in some cases scarcely visible, being little more than a rudimentary representative of the epithelium of the tubes. In accordance with this, I have observed that when the tubes are completely gorged with oil, the Malpighian capsules are either entirely free from this material, or they contain only a few particles scattered over their interior: in some cases, one or two epithelial cells may be quite full, but I never observed a complete engorgement of the Malpighian capsule, and I believe it may be stated in general terms that the Malpighian bodies are the parts of the secretory apparatus, in which the deposit is least abundant in cases of Bright's disease. The accumulation of fat within the capsule does not appear to attain such a degree as to exert destructive pressure on the vessels of the Malpighian tuft.

It may now be well to inquire how far the changes which the kidney undergoes during the progress of Bright's disease, and the symptoms by which these changes are attended, admit of explanation.

It will be necessary to consider for a moment the manner in

which the vessels are arranged in the kidney, and the nature of the Malpighian bodies, as demonstrated by Mr. Bowman, in his admirable paper, "On the Structure and Use of the Malpighian Bodies of the Kidney."* The arrangement of the vessels in the kidney, and the circulation through the gland, may be thus briefly described: a small terminal twig of the artery pierces the dilated extremity of the urinary tube: within the capsule thus formed by the expansion of the tube, the artery breaks up into a capillary plexus, which Mr. Bowman has named the *Malpighian tuft of capillaries*; these capillaries again unite into a single *efferent* vessel, which passes out through the capsule, and goes to form another plexus, which immediately *surrounds the urinary tubes*. The course of the circulation then is from the artery into the Malpighian plexus, which lies *within* the dilated extremity of the urinary tube, and from this plexus through the efferent vessel into the capillary plexus, which lies *external* to the tubes amidst their coils and convolutions.

Any one having a clear conception of the anatomy of the kidney cannot fail to perceive the effect upon the circulation through the gland, which must result from the changes which I have described as primarily and essentially constituting Bright's disease of the kidney. The fat accumulates in the epithelial cells to such an extent as to produce engorgement and dilatation of the cells, and of the tubes which are lined by them; the consequence is, compression of the capillary plexus surrounding the tubes, giving rise to congestion of the Malpighian plexus. This passive congestion of the Malpighian plexus leads to transudation of the serum of the blood, and sometimes to rupture of the delicate vessels of the plexus, and the consequent escape of the colouring matter and fibrin of the blood. These constituents of the blood pass into the tubes, and so become mixed with the urine. Their escape from the blood-vessels is the result of a mechanical impediment to the return of the blood consequent on

* Philosophical Transactions, 1842.

compression of the veins by an accumulation of fat in the tubes.

The influence of a mechanical impediment, in giving rise to the escape of serum and of blood, and their appearance in the urine, is admirably shown by the ingenious experiments of Dr. Geo. Robinson, the details of which have been communicated to this Society, and published in its Transactions.* Dr. Robinson tied the renal vein in rabbits, the result of which was the appearance of albumen and of blood in the urine; the same consequences followed the partial or slow obliteration of the vein.

It is important here to remark, that a mechanical obstacle to the circulation of the blood through the heart or lungs, may give rise to venous congestion of the kidney, and the consequent presence of serum and of blood in the urine. I have had an opportunity of examining the kidneys of two subjects, in both of whom there was albuminous urine, and in one case dropsy during life. In neither case was there a trace of any organic disease of the kidney, but in both cases there was cardiac disease and great congestion of the lungs. In one case, the medullary portion of the kidney was slate-coloured from congestion, and this congestion extended down the ureter, clearly proving the cause of it to be external to the kidney: the Malpighian tufts were also much congested.

The red spots visible on the surface of the kidney in some cases of Bright's disease, and which have been erroneously supposed to be dilated Malpighian bodies, were first shown by Mr. Bowman to be "nothing less than the convolutions of a tube filled with blood, that has burst into it from the gorged Malpighian tuft at its extremity."—(See fig. 15.)

The mechanical compression of the vessels and the impeded circulation of the blood is the cause of the tortuous, dilated, and varicose condition of the veins and arteries which is often seen on the surface of the kidney, and which is well represented in the first of Dr. Bright's plates.†

* Vol. xxvi. † Medical Reports, vol. i.

Another proof of the compression to which the vessels are subjected during the progress of Bright's disease, is the difficulty of injecting the cortical portion of the gland, and the small number of vessels, and of Malpighian bodies, which are seen when thin sections are examined with the aid of a microscope. In fact, the vessels become compressed, and many of them entirely obliterated; and here we have an explanation of the wasting which the kidney undergoes in advanced stages of the disease.

There is a remarkable difference in the effect produced upon the two organs—the kidney and the liver, by an accumulation of fat in their substance; the function of the kidney becomes seriously interfered with, and its nutrition impaired, while the liver appears to suffer but little, either in its nutrition or its functions. In order to explain this diversity, it is sufficient to consider attentively, and compare, the anatomy of the two organs.

The explanation which Mr. Bowman has given of the comparatively little influence which the fat in the hepatic cells exerts upon the circulation through the gland, is probably the true one. The secretory cells of the liver seem to be, as it were, packed in the meshes of the capillary vessels, so that the engorgement of the cells by fat may produce great enlargement of the liver, without any material change in the relative position of the cells and vessels.

In the kidney, the arrangement of the vessels is such, that they must inevitably suffer serious compression from any distension or bulging of the tubes. Each convoluted tube, with its external plexus, and the Malpighian plexus contained within its dilated extremity, may be looked upon almost as a separate gland; each Malpighian tuft being, as Mr. Bowman has shown, a terminal isolated part of the arterial tree. The dilatation of a single convoluted tube might so compress the plexus by which it is surrounded, and thereby so much oppose the escape of blood from the Malpighian plexus, as to give rise to the bursting of its delicate vessels, in consequence of which the supply of arterial blood to the tube would be

cut off; and a less degree of congestion might so far diminish the direct supply of blood, as to lead to the atrophy of the tube in which the obstruction originated.

Another peculiarity in the structure of the kidney, which, doubtless, contributes to the destructive influence of dilatation of the tubes upon the blood-vessels of the organ, consists in the presence of a fibro-cellular matrix, in the form of a regular net-work, the meshes of which have a circular outline (see fig. 14). In the smaller spaces the tubes are packed, each tube in its course being surrounded by many of these fibrous rings, while in the larger spaces are contained the Malpighian bodies. The vessels constituting the "portal plexus" of the kidney are contained in the substance of this fibro-cellular tissue. Since the tubes naturally fill very accurately the spaces in which they are contained, it is evident that a dilatation of the tubes will lead to compression of the fibro-cellular net-work which surrounds the tubes, and of the vessels contained within this tissue. This tissue presents a resisting point, against which the distending force within the tubes may act, and the dilated tubes become constricted, as it were, by ligatures of fibro-cellular tissue, the vessels contained in the substance of the tissue at the same time suffering so much compression that the circulation through them must be greatly retarded or entirely arrested. An obstruction in this part of the circulation re-acts upon the Malpighian tufts; hence congestion of the delicate Malpighian vessels and effusion of serum into the tubes, or even rupture of the vessels and hæmorrhage into the tubes, and subsequently wasting of the tubes themselves.

Without anticipating what will presently be said of the pathology of Bright's disease, I will here offer a few remarks on its stages and forms; and, first, I will venture to assert that there is no inflammatory or congestive stage *preceding* the deposit. The congestion which often accompanies the disease, and which is a consequence of previous morbid changes, may be either active or passive. The way in which passive congestion occurs has already been sufficiently ex-

plained. Active congestion may be thus accounted for:—a large number of the epithelial cells become gorged with fat, and their secreting function is in consequence impaired: those portions of the gland which are less involved in the disease are now called upon to do an increased amount of work; this may lead to active congestion, and the consequent effusion of serum and blood into the tubes. In many cases there probably exists both active and passive congestion of the vessels; but I repeat that this is the *consequence*, and not the *cause*, of the deposit in the gland.

Dismissing, then, a congestive stage, the earliest appearance of fatty degeneration of the kidney would, of course, be recognised by the aid of the microscope before the gland has undergone any change visible to the naked eye. As the accumulation of fat increases, the kidney becomes granular or "mottled" on the surface. The smooth, mottled kidneys are such as have the greater number of the tubes in the cortical portion almost uniformly gorged; the gland is often much increased in size by the great amount of fat in the tubes, the vessels are much compressed, and the surface of the kidney sometimes presents an almost uniform yellowish white colour, with here and there a few vessels which have escaped obliteration. (See 4th plate in 1st volume of Dr. Bright's Reports.) These are generally cases which have run a comparatively rapid course. The secreting function of the kidney has become greatly impaired, and death has been the consequence. The kidney which has arrived at this degree of fatty engorgement, probably never becomes atrophied.

The *granular and atrophied* (Bright's) kidneys are those in which the accumulation of fat takes place less rapidly and uniformly; some convoluted tubes become gorged with fat, forming prominent granulations; and these, compressing surrounding parts, produce obliteration of the vessels and atrophy of the tubes, and thus the entire gland gradually wastes and contracts. These are the cases in which the tubes of the pyramids become filled with fat, part of which, perhaps, has been carried into them from above, while part is contained

in their own epithelium, which, perhaps, (as has already been suggested,) assumes a more active secretory office in consequence of the wasting of the cortical portion of the gland.

I do not maintain that every atrophied kidney, and every kidney presenting a granular appearance, have undergone these changes in consequence of fatty degeneration of the gland; on the contrary, I am well aware that many instances of granular and contracted kidneys are met with, in which the degeneration has been of a totally different kind, and I am also in a position to show that these are not cases of true Bright's disease; that they belong to a class of diseases which the best pathologists have always endeavoured to distinguish from Bright's disease, although, in the absence of any accurate means of definition, diseases totally and essentially different in their nature have often been confounded under one name. (See Appendix, page 22.)

Before speaking of the pathology of Bright's disease, it is important to consider briefly the diseases with which it has been found to co-exist. Dr. Bright appears to attach but little importance to the morbid states of the liver, which he considers to be of comparatively rare occurrence in connection with this form of renal disease; but, on the contrary, the observations of Dr. Christison, M. Rayer, and several other pathologists, have shown that some form or other of liver-disease is a very frequent concomitant of Bright's disease of the kidney. So far as I have been able to ascertain, no pathologist has given any very definite account of the *kind* of liver-disease most commonly associated with Bright's disease.

My own observations have led me to conclude that, in by far the greater number of cases, Bright's disease, or fatty degeneration of the kidney, is associated with a similar fatty degeneration of the liver.

Since the commencement of July, I have made a *post-mortem* examination of 22 cases of Bright's disease of the kidney; and, in 17 of these, there was, in a most marked degree, fatty degeneration of the liver. The liver was com-

monly enlarged, sometimes to a great degree, frequently of a pale yellowish colour, dotted with brown or red; more commonly, however, presenting a nutmeg appearance. Under the microscope, the secreting cells were seen gorged in various degrees with their increased fatty contents; and, in addition, there was much free fat which had escaped from the ruptured cells. (I would here remark, that the quantity of fat in the liver cells, as well as in those of the kidney, varies considerably within what may be considered the limits of health. When I have observed the fat in the liver cells unusually abundant, yet not so much as amounts to complete engorgement of any considerable number of the cells, I have noted the fact, but have not recorded the case as one of fatty degeneracy of the liver.) It has already been stated that there co-existed with Bright's disease, fatty degeneration of the liver in 17 cases out of 22. In 4 of the remaining 5 cases there was a decided increase of fat in the hepatic cells; and in one case only was there no such increase. During the same period I met with only 4 cases of fatty liver which were *not* combined with Bright's disease of the kidney. In 3 of these cases, although there was not a decided fatty degeneration of the kidney, there was still a marked increase of fat in the epithelial cells of this gland. In the fourth case of fatty liver there was *no* increase of fat in the kidney.

Of 23 cases in which both the liver and the kidneys might be considered *healthy*, there was an unusual quantity of fat in *both* organs in 4 cases, in the *liver alone* in 2 cases, in the *kidney alone* in 2 other cases; while, in the remaining 19 cases, the fat existed in what might be considered the usual quantity in *both* organs. In making these observations I have been most careful to avoid every source of error, and I am confident that very similar results will be obtained by every careful observer who will direct his attention to this subject. It is needless to remark how important is a knowledge of these facts in any attempt to explain the pathology of Bright's disease.

It has been fully established by the observations of Dr. Christison, MM. Solon and Rayer, and of many subsequent

observers, that Bright's disease is very frequently associated with tubercular disease of the lungs. Among the 49 cases from which my preceding observations are drawn, there were 14 in which tubercles were found in the lungs. Of these 14, there were six in which there was decided fatty degeneration of both the liver and the kidneys. In one case the *liver alone* was fatty, in one the *kidneys alone*, and, in the remaining 6 cases, there was either *no* increase of fat in the liver or kidneys, or the increase was not so great as to be considered morbid; so that, out of 21 cases of fatty degeneration of the liver, 6 only occurred in connection with tubercular disease of the lungs, while 17 occurred in combination with fatty degeneration of the kidney. The number of observations, I am aware, is too small to enable one to state exactly in what proportion of cases the different combinations to which I have alluded may be expected; but I feel assured that future observers will confirm my own conclusion that fatty degeneration of the liver in different degrees is more commonly associated with Bright's disease of the kidney than with tubercular disease of the lungs.

It has been shown by Dr. Bright and other observers, that, in subjects who die of Bright's disease, the arteries are very commonly affected by those changes to which the terms *atheroma* and *steatoma* have been applied, and which Mr. Gulliver has shown to be a fatty degeneration of the vessels, commencing apparently in the lining membrane, and extending to the deeper tissues.* According to my own observations, it rarely happens that a patient dies of Bright's disease of the kidney without presenting more or less of this fatty degeneration of the arteries. In some cases the degeneration is confined to a few small opaque whitish or yellowish patches in the aorta, while, in others, the disease is very extensive, and affects many of the smaller arteries. This fatty degeneration of the arteries is by no means confined to subjects who are affected with Bright's disease; on the contrary, it is in various degrees an extremely common morbid appearance in persons above the age of 30 who die in the London hospitals.

* Med. Chir. Transactions, vol. xxvi.

It is well known that another frequent concomitant of Bright's disease is hypertrophy of the heart, either with or without valvular disease. The condition of the valves which I have frequently found in cases of Bright's disease, as well as in other cases where there has been no history of rheumatic attacks, has been precisely the same fatty degeneration as occurs in the arteries. Opaque yellow thickening commencing in the investing membrane, and involving the deeper fibrous parts; and this, when examined microscopically, is found to consist entirely of fat, part of which is in the form of free oil globules, while part is contained in cells.

It has been too much the custom to consider all cases of valvular disease, occurring in the earlier periods of life, as the result of rheumatic inflammation of the endocardium. It is most important to be aware that the investing membrane of the valves is liable to the same fatty degeneration as that of the arteries; that the tendency to this disease probably increases in advanced life, but that, in certain conditions of the system, it may occur in the earlier periods of life as one of the consequences of disordered assimilation.

There is one circumstance which Mr. Gulliver does not mention in his paper. I allude to the fact that a certain quantity of fatty matter, varying considerably in different cases, exists constantly in the lining membrane of the *healthy* arteries as well as in that covering the valves. It was not until I had examined the arteries and valves in a considerable number of subjects that I became aware how much fat might exist in these parts without any morbid appearance visible to the naked eye. I think it by no means improbable that, in some of these cases, in which the microscope detects an unusual amount of fat, but which is not sufficiently abundant to constitute any decided morbid appearance visible to the naked eye, this circumstance may be considered an indication of a general perverted nutrition of the artery, by which, probably, its elasticity is impaired. And perhaps in this way may be explained some of those cases of hypertrophy and dilatation of the heart which are not accounted for by the

existence of any visible obstacle to the circulation either at the orifices of the heart or in the course of the arteries.

Let us now look back upon the ground we have gone over. We have seen that, in subjects who die of Bright's disease of the kidney, there is usually found a similar disease in the liver, and, in many cases, in the arteries and on the valves of the heart; the disease being in every case an increase and an accumulation of a material which exists in small quantities in the healthy condition of these parts.

In any attempt to explain the *pathology* of these diseases, their source must be looked for in the processes of digestion and assimilation. The processes of primary or secondary assimilation, or both, fail with regard to this fatty matter, which, not undergoing the changes requisite for its ready elimination from the system, or for its application to the nutrition of the tissues, is thrown into the circulation. An effort is made to carry it off by the liver and kidneys; the fat finds its way into the secreting cells of these glands; its escape from these parts, in a free state, is a slow and uncertain process, and, finding no material in sufficient quantity with which to pass off in a state of combination, the fat accumulates in, and obstructs, the glands.

The increased amount of fat in the secreting cells of the glands must certainly be looked upon as an *effort* to carry off this material. It must also be looked upon as, in a great degree, an *unsuccessful effort*. It will presently be shown that the quantity of uncombined fat in the urine in cases of Bright's disease is seldom greatly increased. As far as regards the result of her effort, then, Nature is as unsuccessful in her attempt to carry off the fat by the glands as to remove it by throwing it into the arteries. In both cases the fat is thrown out of the circulation, but its accumulation in the glands and arteries leads to a serious interference with the functions of these parts.

The conditions under which these diseases occur may be looked upon as analogous to those which give rise to diabetes. In diabetes, in consequence of imperfect digestion or mal-assi-

milation, sugar is eliminated in various exeretions, but especially in that of the kidneys. Again, in the eases in whieh fatty degeneration of the liver and kidneys oecurs, an effort is made to eliminate fat; the sugar being soluble, is readily earried off; the fat being insoluble, and eonsequently diffieult of elimination, aeeumulates in the seereting eells of the glands.

Most pathologists are agreed as to the eonditions whieh favour the development of Bright's disease. It is known to be mueh more eommon in large towns than in the eountry, and in large towns it is mueh more prevalent among the intemperate, ill-fed, and ill-elad inhabitants of cellars, and other imperfeetly aired and lighted apartments, than among those who enjoy more of the eomforts of life. I am indebted to my friend, Mr. Simon, for the opportunity of mentioning an interesting example of the disease artifieially produeed in one of the lower animals. In the eourse of some experiments on the artifieial production of serofulous diseases in the lower animals, Mr. Simon inspeeted the body of a eat whieh had died after having been kept for about six weeks in a dark cellar. On examining the kidney, he found that it presented to the naked eye the appearanee of a mottled Bright's kidney, and on plaeing a portion under the mieroseope, he aseertained that the tubes of the eortieal portion of the gland were eompletely gorged with fat. The liver eells also eontained a great inerease of fat; but their engorgement with this material was less than that of the kidney eells and tubes.

It is eommonly supposed that Bright's disease may eommenee in an attaek of what has been ealled aeute inflammatory dropsy. But here some eaution is neeessary. After the proofs whieh have been addueed of the eonstitutional nature of the disease, it must be evident, that whatever eireumstanees tend to produee it, must do so by aeting otherwise than locally. It is by disturbing the healthy balanee of the digestive and assimilative proeesses, that the intemperate use of spirits acts as an exciting eause of this renal disease, and not by any local irritating effect upon the kidney or liver.

Exposure to wet and cold, and the consequent suspension of the cutaneous functions, may give rise to congestion of the kidneys, scanty, albuminous and bloody urine, and dropsy. But if the patient so attacked was previously of sound constitution, and if he be treated actively, with a view to restore the functions of the skin, to relieve congestion of the kidneys, and to carry off the accumulated fluid by the bowels, the dropsy and the other symptoms will disappear, and the patient will be restored to perfect health. Such an attack has no tendency to terminate in Bright's disease, which, it cannot be too often repeated, is not primarily a disease *in* the kidney, but a constitutional disease, manifesting itself *at* the kidney.

It is very generally believed that Bright's disease may have its origin in an attack of dropsy supervening upon scarlatina. It seems to me that this notion must be accepted with some modification. I have lately had an opportunity of examining the kidneys of three patients who died after an attack of scarlatina. In two of these cases there was albuminous urine, and in one there was dropsy, but in no case did the kidney present any of the characters of Bright's disease. In one, there were no morbid appearances whatever; in a second, there were unequivocal *products of inflammation*; and, in the third, there was great congestion, with blood in the tubes, and other appearances which seemed to indicate an increased amount of functional activity in the organ.

The conclusion which I draw from the preceding facts is, that the dropsy which supervenes upon scarlatina does not depend on Bright's disease, and that if scarlatina ever leads to the development of Bright's disease, it must be through the medium of those constitutional disturbances which would probably at the same time give rise to a similar disease of the liver. I look upon the dropsy occasionally arising during or after an attack of scarlatina, as the result, *partly*, of the cutaneous disease, but *chiefly* of some *materies morbi*, striving for elimination by the kidneys no less than by the skin; and which, in its passage through the former organs, acts as an

irritant, giving rise to active congestion of the kidneys, and the consequent effusion of serum and blood into the urinary tubes.*

Some pathologists entertain the notion that the cardiac disease, which is so commonly associated with the renal, precedes the latter, and so, by inducing congestion of the kidney, gives rise to Bright's disease. This idea will probably prove to be an erroneous one. It is supposed by others that the valvular disease originates in the morbid and irritating condition of the blood consequent on the impaired function of the kidney. It is not improbable that some of the changes which the valves undergo may have their origin in this condition of the blood; but, as has already been mentioned, the condition of the valves which appears to be most commonly associated with Bright's disease, is that of fatty degeneration, which is probably a result of the same common condition as that which gives rise to the renal disease.

I will here offer a few remarks on the microscopical characters of the urine in health and disease.

The presence of fat in the epithelium of the healthy kidney would naturally lead us to expect its occasional appearance in the urine. The existence of oil in healthy urine has probably been noticed by many observers. It is mentioned by Henle. Professor Miller assures me that he

* Since the above was written, I have had an opportunity of examining some well-marked specimens of renal disease occurring as a consequence of scarlatina. The result of these examinations quite confirms me in my opinion that the disease is essentially distinct from Bright's disease; that it is, in fact, an inflammation of the kidney, excited, like the inflammation of the skin which constitutes the eruption of scarlatina, by the passage through the part, of the peculiar fever poison; and as the inflammation of the skin terminates in an excessive development of epidermis and a desquamation of the surface, so the inflammation of the kidney excites an increased development of the epithelium which lines the urinary tubules; this material partly accumulates in, and chokes up, the tubes, while part of it becomes washed out with the urine, and may be detected in large quantities in that fluid by the aid of the microscope. The particulars of this and of other forms of acute and chronic inflammation of the kidney, I purpose to make the subject of a separate communication.

has seldom or never found it entirely absent. I have repeatedly noticed it in the urine of persons whom I had every reason to consider free from renal disease. I believe, however, that its presence in healthy urine will be found not to be a constant occurrence.

It is not improbable that a certain proportion of fatty matter, in a state of combination with some other material, is constantly eliminated from the healthy kidney. The existence of fatty matters as a normal constituent of the urine, is cursorily alluded to by Berzelius, Simon, Scharling, and other chemists, but it has not received that degree of attention which its importance appears to demand.

In Bright's disease, the urine occasionally contains a great increase of fat, but this is by no means a constant, nor, I believe, a very common occurrence. Simon, in his "Chemistry of Man," states that in some chronic cases of Bright's disease, the urine is turbid from the presence of fat.

It is well known that Dr. J. Franz Simon first directed attention to some cylindrical bodies which he had observed in the urine of patients affected with Bright's disease. These bodies are without doubt coagulated fibrin, which has become moulded into the urinary tubes. They often contain blood-discs, nuclei, and fragments of epithelium, and some of them, as I have repeatedly seen, entangle oil globules and cells, containing variable quantities of fat (see figs. 10, 11, and 16, which represent these fibrinous casts).

Epithelial cells containing fat are frequently seen in the urine, even when the fibrinous casts are absent (see figs. 12 and 13). These cells have evidently been washed from the urinary tubes by the current of urine, and their presence in the urine may be looked upon as one of the most certain signs of the existence of Bright's disease. Their diagnostic value is evidently greater than that of the fibrinous casts, since the latter merely indicate a hæmorrhage from the urinary tubes, which may result from more than one disease of the kidney, or from conditions altogether unconnected with renal disease; whereas, the presence in the urine, of epithelial

eells gorged with fat, indieates a eondition of the kidney which is known to be eharaeteristie of Bright's disease.

After Mr. Simon had aseertained that the eat whieh had been eonfined in the eellar was affeeted with Bright's disease, I assisted him in examining, from time to time, the urine of two other cats whieh had also been subjeeted to long eonfinement. In our first examinations we found that the urine of both animals eontained a eonsiderable quantity of free oil globules, as well as epithelial eells enelosing various quantities of oil, some eells being eompletely gorged. At this time there was no albumen in the urine of either animal; but we found, from time to time, that the fat in the urine of one was diminishing, until, at the end of three weeks, it was very small in quantity: and now, on the applieation of heat and nitric aeid, the urine beeame turbid from coagulated albumen. The animal was then killed, and its kidneys were found in an advaneed stage of Bright's disease. There was no deeided inerease of fat in the liver.

The fat in the urine of the other eat eontinued abundant a few days after the death of its fellow, when it suddenly decreased, and some inflammatory produets appeared in the urine. The animal was killed: the kidneys and liver were extremely fatty; the mueous membrane of the bladder was softened, and apparently ulcerated in one pateh. This, evidently, was the souree of the inflammatory produets in the urine, and perhaps of the albumen, whieh appeared simultaneously with the former.

These observations are peculiarly interesting; demonstrating as they do, in a striking way, the eonditions whieh give rise to these fatty degenerations of the liver and kidneys. They are also interesting, on aeeount of the proof which they afford, that the eseape of the serum of the blood with the urine is not an essential eonsequenee of the morbid proeess going on in the kidney; and that it is only when the mechanical obstruetion to the eirculation in the kidney has attained a eertain degree that the eonstituents of the blood eseape with the urine.

There seems reason to believe, that as with the cats, so with the human subject, a greater quantity of oil escapes with the urine during the earlier stages of the disease than in the later periods, when the tubes are more uniformly choked by their accumulated contents; and that in the presence of cells containing enlarged oil globules we shall have a most important sign of the very approach of the disease; one which will give us timely counsel to resort to such means as appear best calculated to arrest the approaching mischief.

In the *treatment* of Bright's disease, with its many complications, it must not be forgotten that the renal disease is a local manifestation of a general constitutional disorder, the removal of which must be attempted, not by the exhibition of violent and depressing medicines, but by strict attention to all those circumstances which are commonly included under the term "hygiene." Pure air, regular exercise, attention to the proper cleanliness and temperature of the skin, with the administration of chalybeate, and such other tonics as circumstances may seem to require—these are the means best calculated to invigorate the system, and so to restore the healthy balance of the functions. In addition, the diet of such patients will require careful regulation; and as a diabetic patient would be cautioned against the use of sugar, so, on the same principle, should the subject of these fatty degenerations be directed to abstain from a fat diet, and from an excessive use of such materials as starch and sugar, which seem difficult of digestion, and which may, perhaps, by a slight chemical change, be converted into fat.

The kidneys will require some special treatment, with a view to relieve congestion, which necessarily interferes more or less with the function of the gland, if it do not increase the tendency to fatty accumulation. The best means of relieving the congested condition of the kidney will be the regulation of the functions of the skin and bowels. Local bleeding may sometimes be called for, and it is a measure often followed by great relief and a manifest improvement in

the secreting power of the kidney; but in the use of this measure we must exercise that degree of caution which is required of us, when we remember the pathological history of the disease with which we have to deal.

I take this opportunity of mentioning that my friend and former fellow-student, Dr. Inman of Liverpool, has compared the specific gravity of the healthy kidney with that of kidneys affected with Bright's disease. The average sp. gr. of the healthy kidney he finds to be about 1046; while in Bright's disease he has found the sp. gr. as low as 1015.

If the object of the preceding pages has been accomplished, I hope to have established, to the satisfaction of the Society, the following points :—

1. That the epithelial or secreting cells of the healthy kidney contain a certain quantity of oil; the proportion of which, under certain circumstances, and within certain limits, may fluctuate considerably.

2. That it is an excessive increase of this fat, leading to engorgement of the epithelial cells, and of the urinary tubes, which constitutes primarily and essentially Bright's disease of the kidney.

3. That the presence of albumen and blood in the urine, and the wasting of the tissue of the kidney, are secondary phenomena, dependent on the mechanical pressure of the accumulated fat.

4. That in the majority of cases, Bright's disease is associated with a similar fatty degeneration of the liver and arteries, and frequently of the valves of the heart; these diseases being related to each other as joint effects of one common constitutional cause.

5. That probably acute inflammatory dropsy, occurring in a person previously healthy, and the dropsy which occasionally supervenes upon scarlatina, have no necessary connection with Bright's disease of the kidney.

6. That most important evidence of the approach and presence of the renal disease may often be derived from a microscopical examination of the urine, in which will be found

fat in unusual quantity; partly in the form of free oil globules, and partly contained in epithelial cells which have escaped from the urinary tubes.

7. That the insight which we have obtained into the peculiar change which the kidney undergoes in Bright's disease, and the knowledge we possess of the simultaneous occurrence of a similar change in other organs, may serve as important guides in the prevention and cure of the disease.

In conclusion, I have to acknowledge my obligation to those gentlemen who have assisted me in obtaining specimens of diseased structure. To Mr. Hewett, of St. George's Hospital, I feel myself peculiarly indebted, for his readiness in giving me access to his admirable record of *post-mortem* examinations made at St. George's, as well as for his kindness in procuring for me, and sending me, specimens.

APPENDIX.

In the preceding communication it will be seen that I have used the terms "Bright's disease" and "fatty degeneration" of the kidney synonymously; and I doubt not that the profession will agree in the propriety of thus restricting the former of these two terms. The term "Bright's disease" has frequently been applied in the most vague and indefinite manner, not only to almost every form of renal disease, but to cases of "albuminuria," quite unconnected with disease of any kind in the kidney. Most pathologists, however, have acknowledged the importance of distinguishing what they have called "true Bright's disease," or "true granular degeneration," of the kidney, from those comparatively infrequent forms of renal disease which they suppose to result from simple inflammation. Rayer believes, that in the presence or absence of albuminuria we have a test by which to dis-

tinguish Bright's disease, or, as he calls it, "albuminous nephritis," from simple chronic inflammation. I believe that Rayer's test will fail in most cases, and that the only one to be relied upon, is the minute anatomy of the diseased product. In the microscope we have a means of distinguishing the fatty from the acute or chronic inflammatory conditions of the kidney, not only after the death of the patient, but in most cases, and with great certainty, during life, and while the disease is in progress.

In conclusion I have to state, that at the commencement of August, when my paper was received by the Society's Secretary, I was not aware that any observations on the minute anatomy of Bright's disease, in any degree resembling my own, had previously been published; nor at the very full meeting of the Society in November, when my paper was read, did any one present appear to be aware of the fact that, so far as regards the mere sight of fat in some cases of renal degeneration, I had been anticipated by more than one observer. The most important observations with which I am acquainted are those of Hecht (de renibus in morbo Brightii degeneratis; Berlin, 1839), Gluge (Anat. Microsc. Unters.; Jena, 1841), Henle (Henle und Pfeufer, Zeitschrift für rationelle Medizim 1842), Canstatt (de morbo Brightii; Erlangen, 1844), and Eichholtz (Müller Archives, 1845; and Medical Gazette, 1845). The above-mentioned authors agree with each other, and with myself, in the simple and very obvious fact, that, in some cases of renal degeneration, fat in large quantities is contained in the substance of the kidney: as to the situation of the fat, and the interpretation of the whole phenomena of the disease, I believe my own views differ essentially from those of any preceding observer.

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EXPLANATION OF PLATE I.

Figs. 1 to 4.—Epithelial cells from a healthy kidney. No. 1 contains no oil; 2, 3, and 4, contain a few small oil globules in their interior. Magnified 400 diameters. See page 2.

Figs. 5 to 8.—Epithelial cells from a kidney affected with Bright's disease. The oil globules in these cells are much larger and more numerous than in those from the healthy kidney. Magnified 400 diameters. See pages 2 and 3.

Fig. 9.—Portion of one of the urinary tubes from a kidney affected with Bright's disease. The oil globules contained in the epithelial cells by which the tube is lined, are here seen through the wall of the tube. Magnified 400 diameters. See page 3.

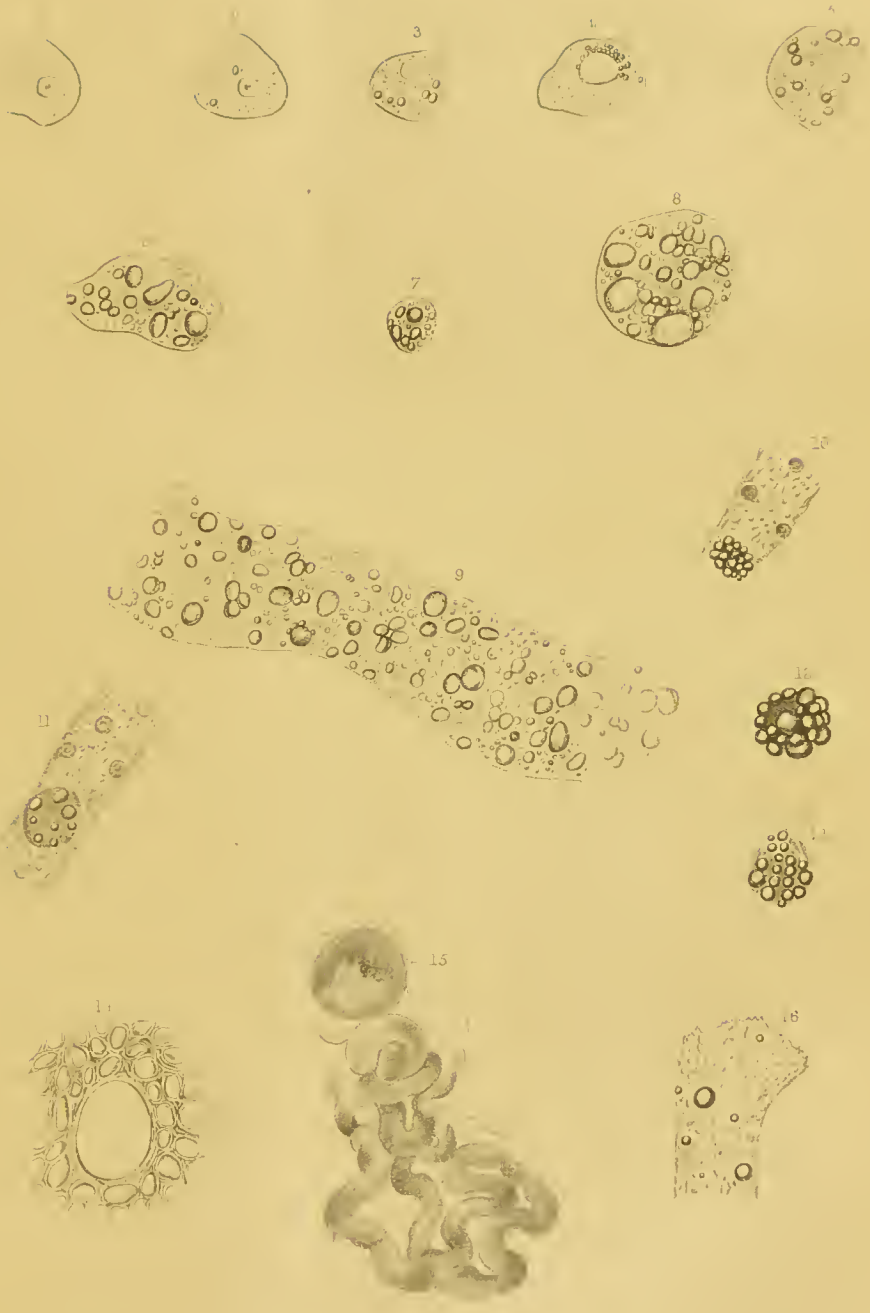
Figs. 10 and 11.—Fibrinous casts of urinary tubes from the urine of a patient labouring under Bright's disease. Each cast entangles blood corpuscles, and a cell having a considerable number of oil globules in its interior. Magnified 200 diameters. See page 18.

Figs. 12 and 13.—Cells containing numerous oil globules from the urine of a patient labouring under Bright's disease. Magnified 200 diameters. See page 18.

Fig. 14.—Fibro-cellular matrix from a healthy kidney, showing one large oval space which contained a Malpighian body, and several smaller spaces of pretty uniform size, in which the convoluted urinary tubes were packed. The substance of the network is made up of fibro-cellular tissue inclosing blood-vessels. Magnified 45 diameters. See page 8.

Fig. 15.—One of the red spots on the surface of a diseased kidney, magnified 45 diameters. A Malpighian capsule and a convoluted tube are seen filled with blood that has burst into them from the gorged Malpighian tuft. See page 6.

Fig. 16.—Fibrinous cast of one of the urinary tubes from the urine of a patient affected with Bright's disease. Several scattered oil globules are seen entangled in the fibrine. Magnified 200 diameters. See page 18.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